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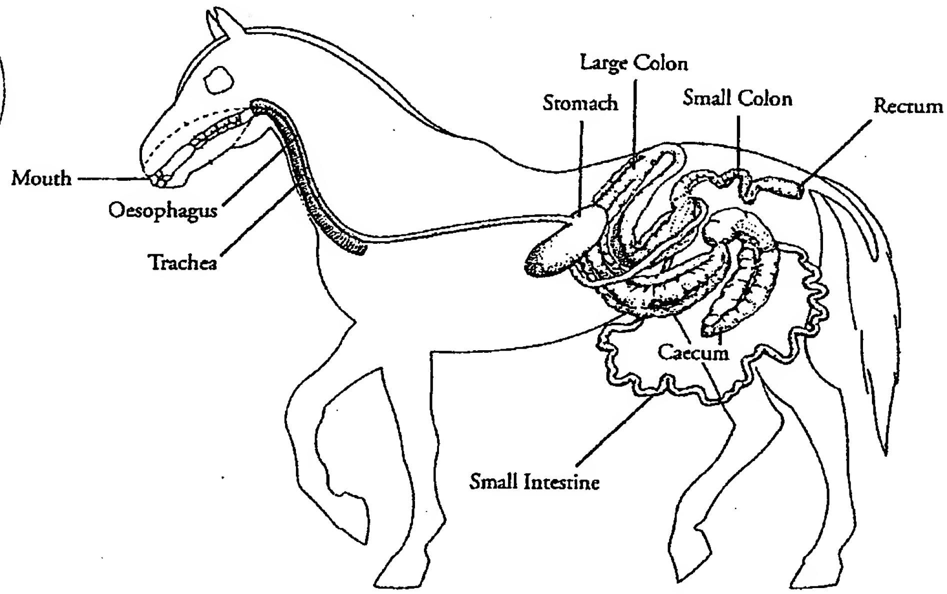

Figure 1.2: The Digestive System of a Horse.

	Volume (%)	Volume (L)	Length (m)	Passage Time	Digestive Activity
Stomach (horses cannot vomit)	8%	7.5-15 L	0.25 m *(20-25cm diameter)*	Water: 75% in 30 mins Dry food: 25% in 30 mins and 98% in 12 hours	Some protein digestion by acid Partial feed breakdown Soaking feed mass with gastric fluid and saliva
Small Intestine	28%	40-50 L	15-22 m *(7-10 cm diameter)*	Water: 2-8 hours Food: 1-8 hours	Major fat and protein digestion Carbohydrate 50-70% Most vitamins and minerals No fibre
Large Intestine Caecum	17%	25-30 L	0.9-1.2 m *(15-25 cm diameter)*	Water: 5 hours Food: 6-12 hours	Fibre 50% residual carbohydrates
Large Colon	34%	50-60 L	3.0-3.7 m *(20-25 cm diameter)*	**Relative passage times** Fresh grass: 24-36 hours	Fibre Water absorption Remaining carbohydrates
Small Colon	11%	18-19 L	3.0-3.2 m *(7.5-10 cm diameter)*	Concentrates: 24-36 hours Pellets: 24-36 hours	Fibre Water absorption
Rectum	2%	2-3 L	0.3 m *(6-7cm diameter)*	Hay: 50-60 hours	Faecal storage Water absorption
Total	100%	143-177 L *(approx. 40 gallons)*	23-31 m *(approx. 100 feet)*	Total transit time: 72-84 hours	

Table 1.3: The major structural sections, capacity and function of the digestive system of a 500 kg horse.

pony can vary markedly with the diet, feeding regime, and time of sampling as well as the site from which samples are obtained. The effect of each of these factors emphasizes the need for clearly defining these parameters.

The pH of stomach contents in ponies fed the control diet (pelleted, hay grain) was near neutrality prior to feeding, but showed a progressive decrease between the 2nd and 8th h. While this could be partially explained by the increase in both VFA and lactic acid concentrations, these reached their highest levels at 4 h, while the lowest pH values were recorded at 8 h. Therefore, the lowest pH values would appear due to HCl secretion.

It is especially interesting to note that the contents of the nonglandular (cranial) and glandular (caudal) portions of the pony stomach registered a similar pH at all times measured and that the lowest values were above those measured in the secretory stomach of man, the dog, or even the cow (9). While the pH measurements of the present study represent a single site and depth within a given segment of gut, the sites were held constant, and this procedure allowed immediate and anerobic measurements of pH. The relatively high pH of the gastric contents would account for the degree of microbial activity, as indicated by the VFA and lactic acid levels. It would also aid in protecting the gastric mucosa from these organic acids, whose potential danger to the tissue at a lower luminal pH has been readily demonstrated by their necrotizing effect on rumen epithelium in cases of acute grain intoxication (16) and by the ulcerogenic effect of 100 mM acetate solutions in the dog stomach (7). The potential danger of VFA would seem to be further compounded by the finding that proper gastric and pyloric mucosa were capable of absorbing VFA from the lumen bath buffered at pH 7.4, but appeared relatively unable to transport these organic acids to the blood side. These studies also showed that the stratified squamous epithelium of the equine stomach, unlike that of ruminant forestomach, appeared quite impermeable to VFA, and its low values for tissue conductance indicated that it was also much less permeable to the passive diffusion of other ions (18).

pH alkali between 3 & 2 18h

The pH of small intestinal digesta tended to increase from approximately 6.0 in the duodenum to 7.3 in the ileum while both VFA and lactic acid concentrations decreased through these segments. The high pH values of ileal contents suggested that the ileum may add a substantial amount HCO_3 (or reabsorb H). In the cecum and large intestine there was a considerable decrease in both the concentrations and amounts of lactic acid while VFA levels increased markedly. Cyclic variations in pH of large intestinal contents between 6.0 and 6.5 appeared to reciprocate with changes in VFA concentration.

Equations developed in studies of the passage of marker through the gastrointestinal tract of these same animals (4) allowed a measure of substrate and VFA entering and leaving the individual segments of large intestine. This information, in turn, allowed calculations of the net production (production minus absorption) of VFA during the intervals between measurements. Figure 6 shows the total quantity and the calculated net appearance or disappearance of VFA for each segment of the large intestine. In all segments, the greatest quantities of VFA were found 8 h and

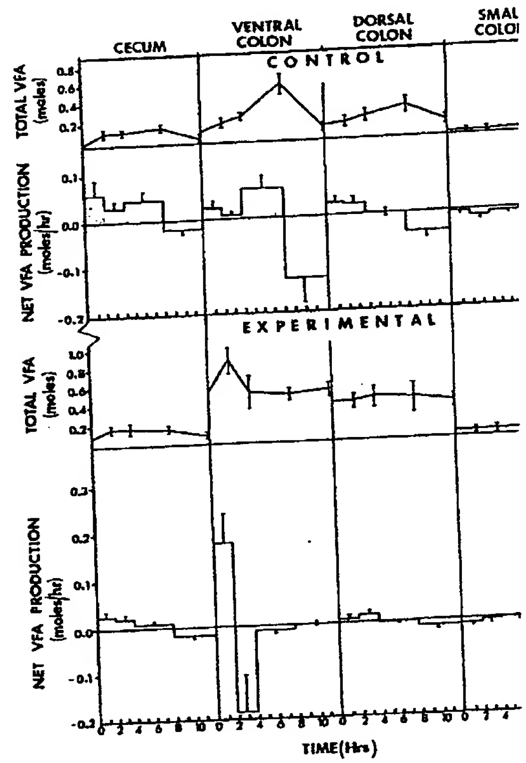

FIG. 6. Content and net production of VFA in segm intestine of ponies fed control and experimental diet. represents mean quality ± SE present at each time of n Outflow of VFA from each segment was determined f fluid passage (4) and mean VFA concentration during ti that were measured. Net production of VFA was then dete net change in VFA content of a given segment between m corrected for amount of VFA entering or leaving segme flow. Standard error for mean values of net VFA producti lated as combined error for fluid passage (4) as well as VFA concentrations and VFA quantities.

the smallest quantities 12 h after feeding the c However, the most rapid net production of VF to occur between 0 and 2 h in the cecum, 4 an ventral colon, and 0 and 4 h (or more, probab in the dorsal colon. The small colon showed n net VFA production. Discrepancies between maximal net VFA production within a segme periods of maximal VFA quantity were due to t the latter was also dependent on digesta passa Periods of net VFA production also correlate maximal rate of appearance of soluble and small markers in each of these segments (4). The acet nate ratios in cecal and ventral colonic digest early digestion of starch followed by the later less soluble carbohydrate, e.g., cellulose (3). uously higher ratio of these two acids in the would indicate a more continuous digestion of carbohydrate.

Results indicated that there was a net a substantial quantities of VFA from the large int the period between 8 and 12 h after feeding, w acids were absorbed more rapidly than they we

From Argenzio et al (1978)

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SOME PHYSIOLOGICAL EFFECTS OF SODIUM BICARBONATE IN DIETS OF YEARLING HORSES[1]

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Summary

A study was made of the physiological effects of adding sodium bicarbonate to growth diets for yearling Quarter Horses. Four treatment groups of four horses each were fed corn-soybean meal-alfalfa based diets for 140 days: (1) control, (2) 1% added $NaHCO_3$, (3) 1% added commercial vitamin-mineral mixture, (4) 1% added $NaHCO_3$ + 1% added vitamin-mineral mixture. Measurements taken every 28 days included individual weights, respiration rates, fecal pH, urine pH; venous pCO_2, pH and packed cell volume; and venous serum sodium, chloride, potassium, calcium, and phosphorus.

Weight changes were not significantly affected by treatments. Increased pH of feeds was associated with increased respiration rates, decreased venous pCO_2, decreased venous pH, increased packed cell volume, increased serum potassium, increased serum phosphorus, and decreased serum chloride. Feed pH had little effect on serum sodium and calcium. Feed pH was positively correlated with urine pH and showed a moderate negative correlation with fecal pH.

Observations suggested that chronic ingestion of 1% sodium bicarbonate was associated with a tendency toward metabolic acidosis, partially compensated by respiratory alkalosis.

Introduction

According to reviews by Baker and Harrison (1979) and Trenkle (1979) some beneficial effects have resulted from the inclusion of sodium bicarbonate in diets of cattle, swine, and poultry. Currently, sodium bicarbonate is being added to some horse diets without researched evidence of its chronic effects.

Acidosis associated with exhaustion was observed in racehorses by Krzywanek (1974). The accumulation of lactic acid may be one of the limiting factors in muscular performance of the horse. Metabolic acidosis also has been associated with severe shock and colic in horses (Rose, 1981). It has been speculated that the addition of sodium bicarbonate to horse diets may ameliorate acidosis.

The purposes of this study were (1) to compare weight changes of yearling Quarter Horses fed similar diets with and without sodium bicarbonate; (2) to assess the physiological effects of chronic ingestion of sodium bicarbonate on the acid-base balance of the horse; and (3) to observe possible interactions between diet supplementation with a commercial vitamin-mineral mixture and addition of sodium bicarbonate.

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Experimental Procedure

Sixteen Quarter Horse yearlings were randomly allotted into four groups of three fillies and one gelding each. All except one purchased filly had similar bloodlines and had been raised in similar environments at the University of Illinois.

Each horse was vaccinated for tetanus and equine encephalomyelitis, and given an intramuscular multivitamin injection providing 1,000,000 U.S.P. units of Vitamin A. Horses were treated for internal parasites just prior to the study and again midway through the study.

All horses were fed the control diet for fourteen days prior to the start of the study (table 1). Thereafter horses were lot-fed their respective test diets for 140 days. Diets were formulated to meet suggested NRC (1978) requirements for yearling horses. Sodium bicarbonate was added at one percent by weight of the total feed to two treatment diets and a commercial vitamin-mineral mixture[1] was added at one percent to two treatment diets, using a 2 x 2 factorial design.

The concentrate portion of the feed was pelletted and was fed at approximately 0900 daily. Hay was fed at 1600. Water was continually available. Test parameters were measured in the morning (0700-1000) prior to feeding, with care taken to minimize excitement and disturbance of horses. Ambient temperatures ranged from 45-85°F (7-29°C) during sampling times during the course of the 140-day study.

Individual weights and respiratory rates were recorded two days apart at the beginning of the study and repeated at 28-day intervals (table 2). Respiratory rates were measured by visual observation of flank movements.

Three venous jugular blood samples were drawn from each horse every 28 days. The first sample of whole blood was collected anaerobically in a heparinized glass syringe, stored in ice, and was analyzed within two hours for pH and pCO_2[2]. Packed cell volume was measured by the microhematocrit technique. The third sample was allowed to clot, centrifuged, and the serum was frozen for subsequent analyses for sodium, chloride, potassium, calcium, and phosphorus concentrations.

Two days after blood collections, individual fecal grab samples and urine specimens were taken and analyzed for pH. To facilitate urine sampling, 2 1/2 cc of furosemide (Lasix) was injected intravenously into each horse.

Feed pellets were analyzed for pH. Digestibilities of test diets are currently being determined.

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[1] Sleek, courtesy of Conagra, Inc., Omaha, NB 68131.
[2] Instrumentation Laboratory Micro 13 Digital pH/Blood gas analyzer.

Results

Feed additives caused marked differences in the pH of the pellets (table 1). The vitamin-mineral pellets (pH 5.780) and the control pellets (pH 6.005) were the most acidic, while the $NaHCO_3$ + vitamin-mineral pellets (pH 7.508) and $NaHCO_3$ pellets (pH7.872) were more alkaline.

Variations in pH of feed were reflected in alterations in several physiological parameters (table 2). Yearlings fed higher pH diets had higher respiration rates. Feed pH was positively correlated with urine pH (corr. coef. = .9723) and was negatively correlated with fecal pH (corr. coef. =-.7588). Venous blood of horses receiving bicarbonate had lower pH and lower pCO_2 values. Average packed cell volumes were greatest for horses fed bicarbonate.

An analysis of venous serum mineral levels showed that increased feed pH was associated with increased potassium, increased phosphorus, and decreased chloride concentrations. Sodium levels appeared unaffected by feed pH but were decreased in the two vitamin-mineral treatments. Calcium concentrations were lower than control values in the $NaHCO_3$ and the vitamin-mineral treatments, while the lowest mean serum calcium level was observed in the $NaHCO_3$ + vitamin-mineral treatment.

Weight gains were not affected by treatments. The larger average daily gain in the vitamin-mineral treatment can probably be attributed to compensatory growth of the purchased filly that began the study in a noticeably thin condition.

Discussion

The high correlation of feed pH with urine pH in this study may be a result of renal compensation for excess absorbed bicarbonate. Elevations in packed cell volume in bicarbonate treatments may be attributed to a diuretic effect resulting in increased water loss in the effort to maintain acid-base balance.

It is thought that the cecum and large intestine of equines are important sites of fiber digestion, and it has been observed that the pH of terminal colon digesta is similer to that of cecal contents (Kern et al., 1974). It may be speculated that the reduced pH of colon digesta in this study may have been associated with a reduced cecal pH, altered microbial populations in the gut and/or reduced digestibility of some feedstuffs. Low fecal pH has been associated with reduced digestibility of starch in cattle fed high concentrate rations (Wheeler and Noller, 1977). Goldberger (1965) suggested that reduced fecal pH may be a result of (1) increased uptake of bicarbonate ions from the gastrointestinal tract, perhaps due to increased activity of carbonic anhydrase; (2) decreased secretion of bicarbonate; and/or (3) increased secretion of hydrogen, chloride, and potassium ions. The altera-in fecal pH of this study, however, were not associated with weight changes. Digestibilities of test diets are currently being determined.

The observed trends in venous pH, pCO_2, and respiratory rate are consistent with a tendency toward metabolic acidosis partially compensated by a respiratory alkalosis (Goldberger, 1965; Rose, 1981). These same trends in venous pH and pCO_2 have been observed by Milne (1974) in horses with a compensated metabolic acidosis during exercise. Chronic ingestion of sodium bicarbonate may thus serve to exacerbate acidotic syndromes during exercise, rather than ameliorate them.

Goldberger (1965) and Rose (1981) suggested that metabolic acidosis may occur as a result of (1) increased production and/or retention of acids normally produced in the body, such as sulfuric, phosphoric, and organic acids; or (2) increased loss of base, such as bicarbonate. Goldberger (1965) further observed that carbonic anhydrase in the kidney tubular cells normally acts to conserve bicarbonate in the body. If the activity of this enzyme is inhibited, large amounts of sodium bicarbonate and water are excreted in the urine. This causes a diuresis and an acidosis, due to the loss of bicarbonate. With a rise in urine pH, an increased amount of potassium is lost in the urine due to an exchange mechanism between potassium and hydrogen ions.

Elevated levels of venous serum potassium in horses are often concomitant with acidosis. Normally, 98 percent of body potassium resides intracellularly. It has been noted that when the pH of extracellular fluids decreases, a shift of potassium ions occurs from intracellular to extracellular space. Rose (1981) observed that, for this reason, plasma potassium values tend not to reflect the total body potassium balance. Potassium depletion may occur even while serum levels are normal or elevated. These observations may account for the elevated serum potassium levels in bicarbonate treatments in this study.

The diuretic effect postulated in this study was likely associated with a loss of cations such as sodium, potassium, and calcium ions in the urine. Goldberger (1965) suggested that a resultant drop in serum calcium would cause an elevation in serum inorganic phosphate levels. The observed increase in serum phosphorus in bicarbonate treatments may be due to these effects. The decreased serum chloride levels in horses receiving bicarbonate may have been due to chloride secretion with hydrogen ions into the gastrointestinal tract or an effort to maintain osmotic balance in the presence of continual loss of cations in the urine.

As excess hydrogen ions from an acidosis situation react with absorbed bicarbonate ions in the blood, carbonic acid is formed. This dissociates into water and carbon dioxide. The respiratory center in the medulla and chemoreceptors in the aortic arch and carotid sinus are sensitive to the partial pressure of carbon dioxide and pH of the blood. Yearlings fed sodium bicarbonate in this study probably increased their respiratory rate in an effort to remove excess carbon dioxide through the lungs. This may have resulted in the decreased venous pCO_2 levels observed in these horses, indicative of a tendency toward respiratory alkalosis.

Further research is needed to elucidate the physiological effects of ingestion of sodium bicarbonate by horses, particularly with reference to exercise physiology.

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TABLE 1. COMPOSITION OF DIETS, AS FED, DAILY

Item	Treatments			
	Control	$+NaHCO_3$	+vitamin-mineral mixture	$+NaHCO_3$ +vitamin-mineral mixture
Pellets, total, kg	10.89	11.23	11.23	11.57
Corn, kg	6.79	6.79	6.79	6.78
Soybean meal, kg	3.52	3.52	3.52	3.52
Dried molasses, kg	.51	.51	.51	.51
Iodized salt, kg	.054	.054	.054	.054
Coloring marker, kg	.011	.011	.011	.011
Sodium bicarbonate, kg	--	.338	--	.337
Vitamin-mineral mixture, kg	--	--	.348	.348
Alfalfa hay, kg	21.77	21.77	21.77	21.77
Total feed, kg	32.66	33.00	33.00	33.34
Sodium bicarbonate, percent of total feed	--	1.02	--	1.01
Vitamin-mineral mixture, percent of total feed	--	--	1.05	1.04
Pellets, pH[1]	6.005	7.872	5.780	7.508

[1]Means of 4 samples per treatment.

TABLE 2. PHYSIOLOGICAL EFFECTS OF SODIUM BICARBONATE AND VITAMIN-MINERAL ADDITION TO DIETS OF YEARLING HORSES

Item	Treatments			
	Control	$+NaHCO_3$	+vitamin-mineral mixture	$+NaHCO_3$ +vitamin-mineral mixture
Average daily gain, kg/day[1]	.287	.287	.353	.287
Respiration rate, min^{-1} [2]	11.7	12.6	11.7	12.1
$\bar{v}$ pH[3]	7.4258	7.4105	7.4356	7.4165
$\bar{v}$ pCO_2, mmHg[3]	49.41	48.33	49.07	47.74
Packed cell volume, %[3]	36.8	39.1	37.5	39.4
Serum sodium, Meg/l[3]	138.9	138.0	136.1	137.9
Serum chloride, Meg/l[3]	97.2	96.6	97.0	96.4
Serum potassium, Meg/l[3]	3.94	3.95	3.80	4.04
Serum calcium, mg/dl[3]	12.00	11.90	11.90	11.62
Serum phosphorus, Meg/l[3]	5.35	5.51	5.34	5.60
Urine pH[3]	7.733	7.861	7.667	7.859
Fecal pH[3]	6.853	6.720	6.790	6.595

[1]Means from 4 horses per treatment over duration of study.
[2]Means of 10 periodic samples from each of 4 horses per treatment over duration of study.
[3]Means of 5 periodic samples from each of 4 horses per treatment over duration of study.

the effect that this can have on metabolism, the immune system, the risk of disease and changes in the skeletal system (see Figure 1). For example the damage to the basement membrane of the hoof lamellae may not be the only basement membrane affected as a result of carbohydrate overload, other systems such as the alveolar tissue in the lungs may be compromised.

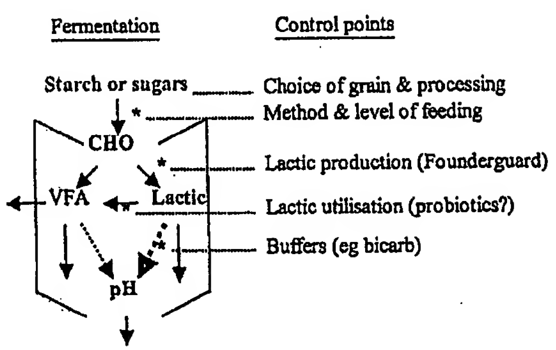
What are the key ways in which to safeguard against acid accumulation in the hindgut?
The control points for preventing lactic acid accumulation through fermentative acidosis in the hindgut are summarised in Figure 2. There are four main ways in which the risk of lactic acid accumulation can be reduced and these are described below.

1 *Feeding management.* Decisions about what grain to feed, how it is processed, how much is fed and in how many meals per day the grain is offered are all under the complete control of the owner or manager. Currently there are no scientifically based guidelines available for the industry and the focus in this paper is to present new information to assist the owner/manager to make the best decisions when preparing to feed grain to horses.

2 *Founderguard.* A narrow range of bacteria (mainly *Streptococcus bovis* and *S. equinus*) are responsible for the conversion of carbohydrate to lactic acid. These bacteria are very sensitive to virginiamycin (Founderguard) and its use to prevent lactic acid accumulation has been very effective in many feeding situations to reduce the risk of laminitis (Rowe et al., 1994) and behavioural changes (Johnson et al., 1998).

3 *Probiotics.* While it is theoretically possible to increase the rate of lactic acid conversion to VFA this requires the delivery of large numbers of viable lactic acid utilising bacteria to the hindgut. In practice this is very difficult to achieve and we are not aware of any probiotic treatment currently available that is likely to be effective in managing the adverse effects of grain feeding in horses.

4 *Buffers and bentonite.* Buffers such as sodium bicarbonate are unlikely to reach the hindgut as the acidic conditions in the stomach convert all bicarbonate to carbon dioxide and water. While bicarbonate may have an effect on the acid/base balance in blood it is unlikely to have any significant effect on fermentative acid accumulation within the gut. Bentonite and other clays are active in reducing the rate of fermentation and can also reduce the severity of loose stools in horses fed grain. However bentonite and other clays do not significantly reduce the risk of lactic acid accumulation and its serious consequences.



Summary of the major pathways of carbohydrate fermentation and acid production and the points at which risk of lactic acid accumulation can be managed.

Selecting suitable grains for horses
Grain-induced laminitis in horses results from overloading the capacity of the small intestine for starch digestion and the subsequent effect that this has on hindgut fermentation. For this reason considerable research effort has focused on measuring small intestine starch digestion in horses fed different grains at varying levels of intake and processed in a number of ways in order to define safe systems for feeding grain (Kienzle et al., 1992), (Meyer, 1993; Meyer et al., 1995a), (Householder et al., 1977), (Arnold, 1982), (Hintz et al., 1971). Estimates of starch digestion in the equine small intestine were obtained by determining the disappearance of starch relative to an inert marker from digesta collected from either fistulated or slaughtered horses. The results are summarised in Figure 3 and show that starch digestibility was highly variable both within grain type and between processing methods. Therefore it is not possible to draw any conclusions from this data with respect to the most suitable grain to feed to horses or how grain should be processed.

More recent studies by Cuddeford (1999), Pagan (1999) and McGilchrist et al (2001) have moved towards techniques for ranking grains and identifying optimal processing methods using indirect measurements. The results of Cuddeford and his colleagues are based on caeca cannulation and the recovery of mobile bags containing grain samples that had been administered directly into the stomach using a naso-gastric tube. Starch digestibility was estimated by measuring the disappearance of starch from the bag. Results from these studies have shown quite clearly the benefits of micronisation in increasing small intestine digestibility of barley starch compared to dry rolled grain. The methods used by Pagan and colleagues are based on the glycaemic response, which is used extensively in human nutrition as a method of determining the effect of starch rich foods on postprandial blood glucose concentrations. Following the consumption of a test diet, blood samples are taken at regular intervals to monitor the rise and fall of blood glucose concentration. The peak glucose concentration and the area under the curve are related to starch digestion and glucose absorption. This technique has been useful in examining the intestinal digestibility of grains i horses (Pagan, 1999) as well as investigating the efficacy of grain processing (McGilchrist 2001). While the glycaemic index measurements are non-invasive and less expensive than measurements involving cannulation and the use of markers to measure digesta flow it is still a relatively expensive and time-consuming measurement. A major disadvantage of in vivo methods is the large variation that occurs between animals in the efficiency of starch digestio and glucose absorption. This variation in starch digestion is not unexpected given that the rat of eating, extent of chewing and also the amount of amylase produced in the intestines of horses has been reported to vary between animals (Meyer et al., 1995). Comparison of results reported from different studies is difficult because the amount and nature of the roughage component of the diet, the level of feeding and the number of meals fed per day varies between studies. Although it is important to account for differences between animals when designing feeding programs it is a source of variation that makes characterization of the diet extremely difficult. In vitro studies offer an alternative to in vivo studies with the advantages of being cheaper and allowing a large number of samples to be compared under a standard se of conditions.

From Rowe et al (2001)
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